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MAXIMUM VOLUNTARY VENTILATION AFTER $+G_x$ IMPACT IN HUMANS

Peter G. Hanson, Captain, USAF, BSC

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6571st Aeromedical Research Laboratory
Aerospace Medical Division
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FOREWORD

This study was conducted during February 1965 by the Biodynamics Branch of the 6571st Aeromedical Research Laboratory.

The author wishes to acknowledge the cooperation and assistance of all members of Dynalectron Corporation, Land-Air Division Aeromed Sub-Group under the supervision of Mr. Mac Connell.

* * *

This technical report has been reviewed and is approved.

CLYDE H. KRATOCHVIL
Lt Colonel, USAF, MC
Commander

ABSTRACT

Eighteen volunteer male subjects were exposed to 20 $+G_x$ impact on the Daisy Decelerator. Measurements of maximum voluntary ventilation (MVV) obtained 10 minutes prior to, immediately after and 20 minutes after impact were compared with previously determined baseline MVV values. The results indicate that MVV performance is elevated immediately after impact. It is suggested that this response is related to subject anxiety with accompanying sympathicotonia.

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I

INTRODUCTION

Exposure to sustained acceleration in the $+G_x$ axis results in pulmonary dysfunction in human and laboratory animal subjects. Significant diminutions in pulmonary function as measured by tidal volume, vital capacity, maximum voluntary ventilation (MVV) and pulmonary diffusion have been demonstrated in humans (Ref. 1). Roentgenographic studies in humans and post-acceleration autopsy findings in dogs also suggest that the anterior-posterior displacement of the pulmonary structures within the thoracic cavity results in the engorgment of pulmonary vessels and atelectasis (Ref. 2, 3). These effects develop after 3 to 10 minutes of acceleration at 6 to 12 $+G_x$ (Ref. 4).

Impact acceleration in the $+G_x$ axis produces a marked anterior-posterior compression of the thoracic cavity and inertial displacement of the cardio-pulmonary organs (Ref. 5) which could result in serious lung damage at high impact levels. Accumulated experience with human test subjects on the Daisy Decelerator (Ref. 6) indicate that no clinically perceptible impairment of pulmonary function occurs after $+G_x$ impact at levels up to 25 G; however, these observations have not been verified with quantitative measurements. Therefore, it was of interest to measure MVV before and after $+G_x$ impact to more clearly evaluate the physiological effects of impact on pulmonary function in humans.

II

METHODS

A. Experimental Design

Experiments were conducted during the months of January and February, 1965. A total of 18 volunteer subjects were exposed to impact on the Daisy Decelerator in the $+G_x$ vector (0-5-180).[1] The conditions of deceleration were: brake entrance velocity 30 fps, onset of deceleration 1000 G/sec. with peak of 20 $\pm$ ¯ G. Each subject was exposed once to the impact conditions.

B. Subject Protocol

A baseline MVV was determined on each subject prior to the actual impact experiments. The measurement technique will be discussed below.

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[1] 0-5-180 refers to the orientation of the subject in degrees of roll, pitch and yaw on a polar coordinate system. In the neutral position of 0-0-0 the subject is seated upright facing forward toward the point of impact. In the 0-5-180 orientation the subject is seated upright, 5 degrees back from perpendicular with the back facing the point of impact. Inertial forces in this orientation are directed from anterior to posterior.

On the day of an experiment, the subject reported to the Daisy Decelerator facility approximately one hour prior to the scheduled impact. After a medical examination and placement of vectorcardiogram leads, a pre-impact MVV was determined. The subject was then taken to the Daisy sled for the impact run. A second pre-impact MVV was determined while the subject sat in the Daisy sled prior to attachment of the restraint harness. Sled firing and impact followed within 10 minutes of this pre-impact MVV.

Immediately after impact, the restraint harness was released and the subject was examined briefly to ascertain the state of consciousness and whether any obvious physical injury had occurred. The immediate post-impact MVV was then measured. An average time of 40 seconds elapsed from impact to the beginning of the MVV determination. Following a post-run physical examination, a final MVV measurement was made. This measurement was approximately 20 minutes post-impact.

C. Determination of Maximum Voluntary Ventilation

Maximum voluntary ventilation is defined as the maximum volume of air which can be moved in and out of the lungs per unit time (Ref. 7). It is usually accomplished by having the subject breathe into a spirometer through a low-resistance respiratory valve. The subject is urged to breathe rapidly and yet deeply to produce the maximum possible ventilation volume. Several trial sessions are required to attain consistent results. Since the over-breathing process produces respiratory alkalosis, the collection period is limited to 10 or 15 seconds.

The conditions of this study required a portable gas collection system so that MVV could be determined on the Daisy Decelerator before and after impact. A 100-liter capacity Douglas bag (Thomas) was fitted with a plastic two-way respiratory valve (Collins No. P-304). This system [illegible] supported by a metal frame so that the bag would hang freely and th[illegible] [illegible]alve remain upright for presentation to the subject (Fig. 1). Prior to [illegible] MVV determination, the bag was evacuated with a Collins motor blower (No. P-533) and the bag neck clamped below the point of respiratory valve insertion with a quick release hose clamp. The valve was then inserted and secured with a hose clamp.

A MVV determination was carried out with the subject seated, nose clamp in place, and the respiratory valve mouthpiece held firmly to prevent expulsion. At the end of a normal expiration, the subject was instructed to begin maximum effort breathing which continued over a timed 15-second period. At the end of 15 seconds the mouthpiece was withdrawn from the subjects' mouth and the bag neck quickly clamped below the valve.

The volume of expired air collected in the Douglas bag was then measured by connecting the bag to the intake end of a Collins motor blower which was in turn connected to a dry gas meter (American Meter Co.) so

Figure 1. Subject and Equipment During MVV Determination

that the contents of the bag were expelled through the gas meter. Gas temperature readings were taken when approximately half of the collected volume had passed through the meter. Barometric pressure was obtained from a mercury manometer situated in the room where the gas volume was measured.

D. Treatment of Data

MVV is usually expressed in liters per minute BTPS. Because of the 4,100 foot altitude of Holloman Air Force Base, volumes measured (ATPS) were relatively large compared to clinical values established near sea level (Ref. 8). The data in this paper, therefore, are expressed in liters per minute (BTPS) corrected to 760 mm Hg. Calculation of MVV was accomplished by the following method:

$$MVV = \dot{V}_E \times 4 \times K \times \frac{(P_B - P_{H_2O})}{760 \text{ mm Hg} - 47 \text{ mm Hg.}} \times \frac{273^\circ + T_B}{273^\circ + T}$$

Where:

MVV = maximum voluntary ventilation (liters/minute BTPS)

$\dot{V}_E$ = expired volume for 15 seconds (liters ATPS)

K = dry gas meter correction factor

P_B = ambient barometric pressure (mm Hg.)

T = gas temperature ($^\circ$C) in gasometer

P_{H_2O} = vapour tension of water at temperature of gasometer.

T_B = body temperature (37°)

III

RESULTS

The baseline and predicted MVV values along with the physical characteristics of each subject are presented in Table I. With the exception of several severe discrepancies, the measured baseline values are within 15 percent of the predicted values. The comparatively lower volumes obtained in the baseline determinations may be partially attributed to the sitting position of the subjects in this experiment. Predicted values were determined in the standing position (Ref. 9),

Table I. BASELINE AND PREDICTED MVV VALUES

						MVV (L/MIN BTPS)	
	Subject	Age	Ht.(cm)	Wt.(kg)	S.A.(M^2)	Baseline ± $S\bar{x}$	Predicted*
1.	J.L.A.	30	170	71	1.81	161 ± 1.1	148
2.	C.V.A.	27	170	59	1.68	129 ± 5.7	141
3.	J.S.A.	23	183	88	2.10	145 ± 1.1	181
4.	W.M.A.	35	183	89	2.14	133 ± 8.1	171
5.	J.R.B.	24	178	59	1.74	163 ± 5.3	148
6.	G.T.B.	28	190	84	2.20	162 ± 3.5	183
7.	W.C.C.	25	170	87	1.99	164 ± 1.9	168
8.	W.E.	26	183	83	2.10	136 ± 3.7	176
9.	J.O.E.	23	181	75	1.94	126 ± 4.3	165
10.	J.F.H.	30	178	79	1.98	133 ± 7.8	162
11.	K.D.H.	33	185	96	1.98	133 ± 8.2	158
12.	C.W.H.	24	183	74	1.99	174 ± 6.0	168
13.	A.A.P.	31	183	87	2.15	174 ± 6.7	173
14.	J.L.P.	22	178	79	1.99	148 ± 3.2	170
15.	J.W.P.	27	178	87	2.15	178 ± 4.3	178
16.	J.W.R.	23	181	75	1.98	118 ± 3.7	170
17.	L.M.S.	24	178	71	1.88	162 ± 0.1	160
18.	J.P.W.	33	183	79	2.20	153 ± 2.4	178

*Motley, H.L.: J. Chest. Dis: 24, 378, 1953.

MVV = (97 - 0.50 Age (yrs)) x S.A.(M^2) L/MIN (BTPS).

which is known to yield greater MVV volumes than the recumbent or sitting position. Reproducibility of the MVV measurement normally increases with repeated trials (Ref. 10). In our determinations an average of five trials were necessary to establish an acceptable baseline which was set at $|S\bar{x}| \leq 10$ L/min. This figure agrees with obtained values in multiple determinations as discussed by Bartels et al. (Ref. 10).

The results of the experimental conditions are summarized in Figure 2. These data represent three groups of MVV measurements obtained; one preceding and two following impact, each plotted as a function of the baseline MVV. Comparison of the groups is made to illustrate the major trends observed in these experiments. In general, there is an increased MVV 10 minutes prior to impact compared to baseline measurements (B_2 vs B_1). Immediately after impact the elevation in MVV is more pronounced (P_1 vs B_1). Twenty minutes after impact there is a diminished MVV in seven subjects, no change in five and continued elevation in the remaining six.

The changes observed for individual subjects are tabulated in Table II along with a statistical summary of the group data. Interindividual variability clearly accounts for the rather small alteration in MVV when expressed as group averages. A statistical analysis based on Wilcoxons' matched-pairs signed-ranks test (Ref. 11) has been included to evaluate the magnitude and direction of the observed differences. The increased MVV immediately after impact (P_1) is significantly ($P \leq .05$) greater than the baseline (B_1). Changes observed in the other comparisons are not significant.

IV

DISCUSSION

Several factors contribute to subject performance during MVV measurement: the muscular force available, the co. liance of the lungs and thoracic cage, and resistance of the airway and pulmonary thoracic tissues (Ref. 8). Therefore, the MVV measurement does provide a useful overall evaluation of breathing mechanics. However, changes in MVV also may reflect a combination of these factors which precludes a definitive diagnosis without ancillary studies of pulmonary function.

The results of this study indicate that $+G_x$ impact ($20 \pm 1\ G_x$) produces no pulmonary dysfunction as measured by MVV. The data actually suggest that prior to and immediately after the impact exposure subject MVV is elevated above baseline values. This phenomenon may be caused by progressive anxiety which is clearly observable in most human subjects prior to impact testing. After the impact episode, subjects tend to relax and anxiety subsides. Increased secretion of epinephrine, which accompanies anxiety, could produce bronchodilation and increased voluntary

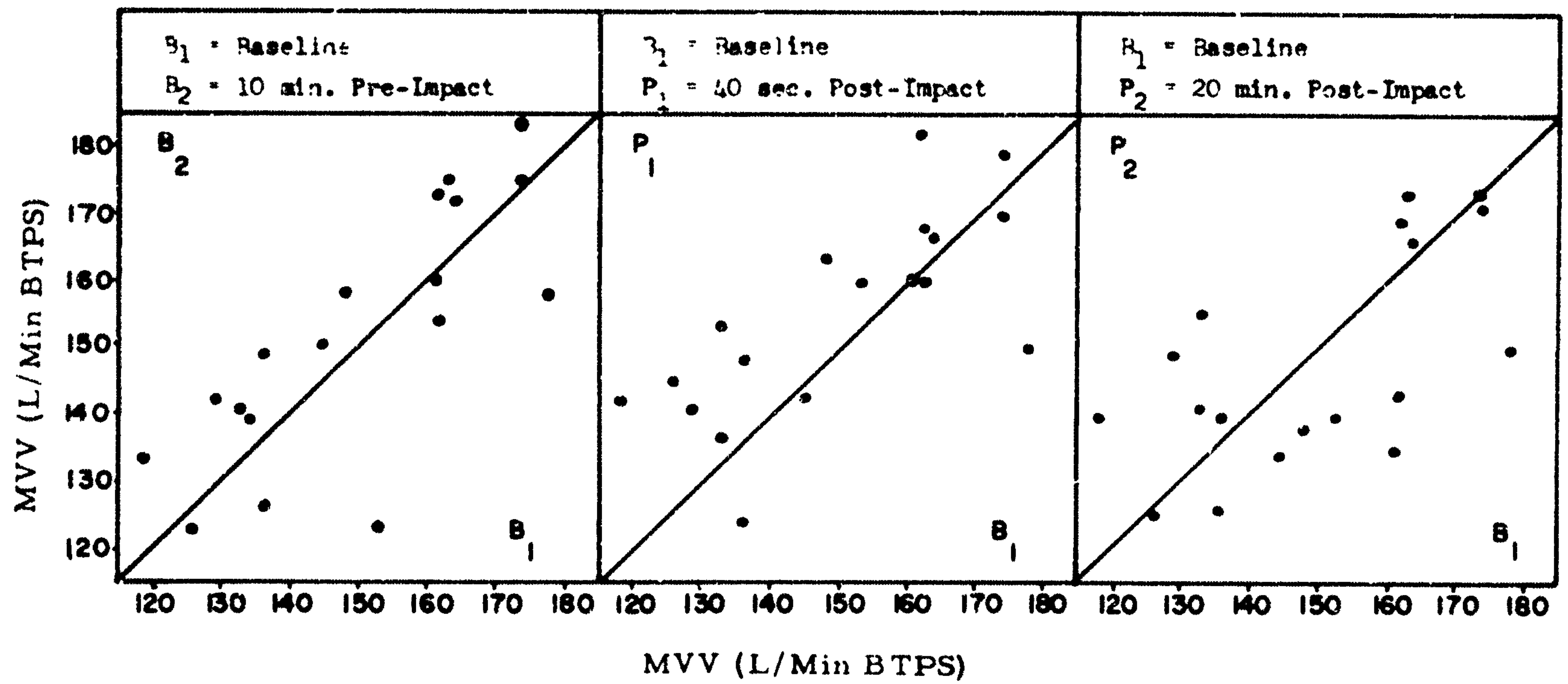

Figure 2. Combined Group Data for MVV Measurements Prior to and After Impact

Table II. STATISTICAL SUMMARY OF EXPERIMENTAL RESULTS

Changes in MVV L/Min BTPS

	Subject	$(B_2 - B_1)$ d	$(B_2 - B_1)$ rank	$(P_1 - B_1)$ d	$(P_1 - B_1)$ rank	$(P_2 - B_1)$ d	$(P_2 - B_1)$ rank
1.	J.L.A.	1	1.5	0		-26	-14.0
2.	C.V.A.	13	13.5	12	10.0	20	11.0
3.	J.S.A.	5	4.0	-2	-1.5	-6	-4.0
4.	W.N.A.	6	5.0	4	4.5	8	6.0
5.	J.R.B.	12	12.0	5	6.5	11	9.0
6.	G.T.B.	16	16.0	20	14.5	7	5.0
7.	W.C.C.	8	7.5	3	3.0	2	1.5
8.	W.E.	10	-10.0	-12	-10.0	-10	-7.5
9.	J.O.E.	-3	-3.0	19	13.0	0	
10.	J.F.H.	7	6.0	20	14.5	22	12.5
11.	K.D.H.	13	13.5	12	10.0	4	3.0
12.	C.W.H.	1	1.5	5	6.5	0	
13.	A.A.P.	10	10.0	-4	-4.5	-2	-1.5
14.	J.L.P.	10	10.0	16	12.0	-10	-7.5
15.	J.W.P.	-20	-16.0	-28	-17.0	-28	-15.0
16.	J.W.R.	15	15.0	25	16.0	22	12.5
17.	L.M.S.	-8	-7.5	-2	-1.5	-19	-11.0
18.	J.P.W.	-25	-17.0	7	8.0	-13	-10.0

$\bar{X} = 2.8$ $T = -53.5$ $\bar{X} = 6.1$ $T = -34.5$ $\bar{X} = -1.0$ $T = -70.5$

$S\bar{x}_D = \pm 11.5$ $P = N.S.$ $S\bar{x}_D = \pm 12.1$ $P \leq 0.05$ $S\bar{x}_D = \pm 15.5$ $P = N.S.$

muscle activity and thus improve the MVV performance (Ref. 10). The greatest increase in MVV occurs immediately after impact exposure when the subject has sustained the maximum anxiety. Urinary excretion of catecholamines, as measured by vanilmandelic acid (VMA), has been studied under similar impact conditions and is known to increase in urine specimens collected 10 minutes prior to impact (Ref. 12). The combination of these observations suggest that increased adrenergic activity in the subjects prior to impact exposure may account for the elevated MVV. Decreased MVV which occurs 20 minutes after impact is not significant and probably reflects the more relaxed attitude of the subjects.

Transient displacement of the pulmonary tree which occurs during $+G_x$ impact does not produce a measureable change in MVV in contrast to a significant decrease in pulmonary function observed with short-term $+G_x$ centrifugation. The latter changes are thought to be associated with radical alterations in pulmonary blood flow and alveolar ventilation produced by prolonged hydrostatic pressure imbalance within the lungs and thorax (Ref. 2). The average duration of impact G in this experiment was 80 to 100 msec. Hydrostatic pressure changes which occur within this time interval probably do not alter pulmonary circulation sufficiently to produce areas of uneven perfusion.

It is concluded that $+G_x$ impact at the magnitude imposed in these experiments does not produce significant diminution in maximum voluntary ventilation performance. Changes which do occur may tentatively be explained by subject anxiety attendant to impact exposure.

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